Amendments to the Claims

This listing of claims will replace all prior versions, and listings, of claims in the application:

Listing of Claims:

1. (Original) A method for modulating gluconeogenesis comprising contacting a cell with an agent that modulates PGC-1 expression or activity, such that gluconeogenesis is modulated.

2. (Original) The method of claim 1, wherein PGC-1 expression or activity is increased.

3. (Original) The method of claim 1, wherein PGC-1 expression or activity is decreased.

4. (Original) The method of claim 1, wherein gluconeogenesis is increased.

5. (Original) The method of claim 1, wherein gluconeogenesis is decreased.

6. (Original) The method of claim 1, wherein the agent is a PGC-1 nucleic acid molecule.

7. (Original) The method of claim 6, wherein the PGC-1 nucleic acid molecule is derived from a human.

8. (Original) The method of claim 7, wherein the PGC-1 nucleic acid molecule comprises the nucleic acid sequence of SEQ ID NO:4.

9. (Currently Amended) The method of claim ~~8~~7, wherein the PGC-1 nucleic acid molecule comprises the nucleic acid sequence of SEQ ID NO:4, wherein nucleotides 518-532 of SEQ ID NO:4 are deleted.

10. (Original) The method of claim 6, wherein the PGC-1 nucleic acid molecule encodes a dominant negative PGC-1 polypeptide.

11. (Currently Amended) The method of claim 10, wherein the dominant negative PGC-1 polypeptide has a mutated LXXLL motif (SEQ ID NO.:3).

12. (Currently Amended) The method of claim 11, wherein at least one amino acid residue of the LXXLL motif (SEQ ID NO.:3) is deleted.

13. (Currently Amended) The method of claim 11, wherein at least one leucine residue in the LXXLL motif (SEQ ID NO.:3) is substituted with another amino acid residue.

14. (Currently Amended) The method of claim 13, wherein the leucine residue at the fourth position of the LXXLL motif (SEQ ID NO.:3) is substituted with alanine.

15. (Original) The method of claim 6, wherein the PGC-1 nucleic acid molecule is an antisense PGC-1 nucleic acid molecule.

16. (Original) The method of claim 6, wherein the PGC-1 nucleic acid molecule is contained within a vector.

17. (Original) The method of claim 16, wherein the vector is an adenoviral vector.

18. (Original) The method of claim 1, wherein the agent is a PGC-1 polypeptide.

19. (Original) The method of claim 18, wherein the PGC-1 polypeptide is derived from a human.

20. (Original) The method of claim 19, wherein the PGC-1 polypeptide comprises the amino acid sequence of SEQ ID NO:5.

21. (Currently Amended) The method of claim ~~20~~19, wherein the PGC-1 polypeptide comprises the amino acid sequence of SEQ ID NO:5, wherein residues ~~144~~-148 of SEQ ID NO:5 are deleted.

22. (Original) The method of claim 18, wherein the PGC-1 polypeptide is a dominant negative PGC-1 polypeptide.

23. (Currently Amended) The method of claim 22, wherein the dominant negative PGC-1 polypeptide has a mutated LXXLL motif (SEQ ID NO.:3).

24. (Currently Amended) The method of claim 23, wherein at least one amino acid residue of the LXXLL motif (SEQ ID NO.:3) is deleted.

25. (Currently Amended) The method of claim 23, wherein at least one leucine residue in the LXXLL motif (SEQ ID NO.:3) is substituted with another amino acid residue.

26. (Currently Amended) The method of claim 25, wherein the leucine residue at the fourth position of the LXXLL motif (SEQ ID NO.:3) is substituted with alanine.

27. (Original) The method of claim 1, wherein the agent is a polypeptide that binds to
PGC-1.

28. (Original) The method of claim 1, wherein the agent is a small molecule.

29. (Original) The method of claim 1, wherein the interaction between PGC-1 and HNF-4α is decreased.

30. (Original) The method of claim 1, wherein the cell is a hepatocyte.

31. (Original) The method of claim 30, wherein the hepatocyte is selected from the group consisting of a primary hepatocyte and a Fao hepatoma cell.

32. (Original) The method of claim 1, wherein the method is performed *in vitro*.

33. (Original) The method of claim 1, wherein the method is performed *in vivo*.

34. (Original) A method for identifying a compound capable of modulating gluconeogenesis comprising:

a) contacting a cell with a compound; and

b) determining whether PGC-1 expression or activity is modulated.

35. (Original) The method of claim 34, wherein PGC-1 expression or activity is increased.

36. (Original) The method of claim 34, wherein PGC-1 expression or activity is decreased.

37. (Original) The method of claim 34, wherein PGC-1 expression is measured by Northern blotting.

38. (Original) The method of claim 34, wherein determining whether PGC-1 activity is modulated comprises determining whether expression of at least one of phosphoenolpyruvate carboxykinase, glucose-6-phosphatase, and fructose-1,6-bisphosphatase is modulated.

39. (Original) The method of claim 38, wherein expression is measured by Northern blotting.

40. (Original) The method of claim 38, wherein expression is measured by measuring the expression or activity of a reporter construct comprising the promoter/enhancer region from at least one of the phosphoenolpyruvate carboxykinase, glucose-6-phosphatase, and fructose-1,6-bisphosphatase genes, operatively linked to a nucleic acid molecule encoding a reporter gene.

41. (Original) The method of claim 34, wherein determining whether PGC-1 activity is modulated comprises determining whether glucose output from the cell is modulated.

42. (Original) The method of claim 34, wherein the cell is a hepatocyte.

43. (Original) The method of claim 42, wherein the hepatocyte is selected from the group consisting of a primary hepatocyte and a Fao hepatoma cell.

44. (Original) A method for identifying a compound capable of treating a disorder characterized by aberrant gluconeogenesis comprising assaying the ability of the compound to modulate the expression or activity of PGC-1 to thereby identify a compound capable of treating a disorder characterized by aberrant gluconeogenesis.

45. (Original) The method of claim 44, wherein the disorder is a disorder characterized by overproduction of glucose.

46. (Original) The method of claim 45, wherein the disorder is diabetes.

47. (Original) The method of claim 46, wherein the diabetes is selected from the group consisting of: type 1 diabetes, type 2 diabetes, and maturity onset diabetes of the young.

48. (Original) The method of claim 45, wherein the disorder is obesity.

49. (Original) The method of claim 44, wherein the disorder is a disorder characterized by underproduction of glucose.

50. (Original) A method for identifying a compound which inhibits the interaction of the PGC-1 protein with a target molecule comprising contacting, in the presence of the compound, the PGC-1 protein and the target molecule under conditions which allow binding of the target molecule to the PGC-1 protein to form a complex; and detecting the formation of a complex of the PGC-1 protein and the target molecule in which the ability of the compound to inhibit interaction between the PGC-1 protein and the target molecule is indicated by a

decrease in complex formation as compared to the amount of complex formed in the absence of the compound.

51. (Original) The method of claim 50, wherein the target molecule is HNF-4α.

52. (Original) The method of claim 50, wherein the target molecule is the phosphoenolpyruvate carboxykinase promoter.

53. (Currently Amended) A method for treating a subject having a disorder characterized by aberrant gluconeogenesis comprising administering to the subject an agent capable of modulating PGC-1 expression or activity, such that the disorder is treated.

54. (Original) The method of claim 53, wherein the disorder is a disorder characterized by overproduction of glucose.

55. (Original) The method of claim 54, wherein the disorder is diabetes.

56. (Original) The method of claim 55, wherein the diabetes is selected from the group consisting of: type 1 diabetes, type 2 diabetes, and maturity onset diabetes of the young.

57. (Original) The method of claim 54, wherein the disorder is obesity.

58. (Original) The method of claim 53, wherein the disorder is a disorder characterized by underproduction of glucose.

59. (Original) The method of claim 54, wherein PGC-1 expression or activity is decreased.

60. (Original) The method of claim 58, wherein PGC-1 expression or activity is increased.

61. (Original) The method of claim 54, wherein gluconeogenesis is decreased.

62. (Original) The method of claim 58, wherein gluconeogenesis is increased.

63. (Original) The method of claim 53, wherein the agent is a PGC-1 nucleic acid molecule.

64. (Original) The method of claim 63, wherein the PGC-1 nucleic acid molecule is derived from a human.

65. (Original) The method of claim 64, wherein the PGC-1 nucleic acid molecule comprises the nucleic acid sequence of SEQ ID NO:4.

66. (Currently Amended) The method of claim ~~65~~ 64, wherein the PGC-1 nucleic acid molecule comprises the nucleic acid sequence of SEQ ID NO:4, wherein nucleotides 518-532 of SEQ ID NO:4 are deleted.

67. (Original) The method of claim 63, wherein the PGC-1 nucleic acid molecule is an antisense PGC-1 nucleic acid molecule.

68. (Original) The method of claim 63, wherein the PGC-1 nucleic acid molecule encodes a dominant negative PGC-1 polypeptide.

69. (Currently Amended) The method of claim 68 wherein the dominant negative PGC-1 polypeptide has a mutated LXXLL motif (SEQ ID NO.:3).

70. (Currently Amended) The method of claim 69, wherein at least one amino acid residue of the LXXLL motif (SEQ ID NO.:3) is deleted.

71. (Currently Amended) The method of claim 69, wherein at least one leucine residue in the LXXLL motif (SEQ ID NO.:3) is substituted with another amino acid residue.

72. (Currently Amended) The method of claim 71, wherein the leucine residue at the fourth position of the LXXLL motif (SEQ ID NO.:3) is substituted with alanine.

73. (Original) The method of claim 63, wherein the PGC-1 nucleic acid molecule is contained within a vector.

74. (Original) The method of claim 73, wherein the vector is an adenoviral vector.

75. (Original) A compound identified by the method of claim 34.

76. (Original) A compound identified by the method of claim 44.

77. (Original) A compound identified by the method of claim 50.